disappearance of headache. The exaggerated knee jerk on the left side had also disappeared, as had likewise the Babinski sign and the Gordon reflex.

For five days after the operation the patient progressed favorably. He then, however, again became somnolent, together with a prompt return of the Gordon and Babinski signs and a plus knee jerk upon the left side. De Keen decided not to re-open the old flap, but to make a trephine opening upon the opposite side, which was done with a negative result. The patient died a few days later, death being preceded by several left-sided convulsive seizures.

An autopsy revealed a large hemorrhage which had taken place on the right side between the dura and the bone in the osteoplastic flap, besides a second hemorrhage into the substance of the dura, separating it into its two anatomical layers.

The fact that the Gordon sign was so pronounced, the fact that it could be elicited with such ease provided pressure was made over the proper place, and the very evident relation which it bore to the lesion on the opposite hemisphere coming and going, so to speak, with the exudation and hemorrhage, are most interesting. It certainly seems to give the Gordon reflex a distinct semeiological value.

Dr. Weisenburg criticized Dr. Gordon's method in obtaining Oppenheim's reflex. According to Oppenheim, deep pressure should be made along the side of the tibia in a most energetic manner, whereas Dr. Gordon only gently stroked this part. Dr. Weisenburg did not believe that the extensor response of the large toe, as obtained by Dr. Gordon, was different from that obtained by Babinski or Oppenheim. It is well known that any involvement of the pyramidal tracts, due to a central or spinal lesion, will cause a loss of some power in the flexors of the toes. Such patients, when walking or moving their limbs, will constantly have an extensor movement of the large toe, and sometimes of all the toes. In a case of excessive spasticity any voluntary movement of the lower limbs, or irritation of any portion of the skin or of the muscles of these limbs will cause an extension of the large toe, or a typical Babinski response. Any method employed to elicit the extensor response is of some value and should by all means be used, but no distinction should be claimed for any one method, the result of which after all gives similar results.

In answer to Dr. Weisenburg's criticism Dr. Gordon stated that it was his custom to test for the reflex with the hammer, but not having one with him at the time he had used his fingers instead, making deep pressure along the sides of the tibia.

PHILADELPHIA NEUROLOGICAL SOCIETY.

January 23, 1906.

The President, Dr. Joseph Sailer, in the Chair.

A Case of Double Facial Palsy of Cerebral Origin.—This was exhibited by Dr. Maier for Dr. Spiller. The patient, a man sixty-two years of age, had had two apoplectic attacks. His bilateral facial paralysis was of recent development. He could not close his eyelids or wrinkle his forehead well on either side, and had very little voluntary power in the lower distribution of the facial nerve on either side, but a little more on the right than on the left. Faradic irritation of the seventh nerve or of the muscles of this distribution gave prompt response on each side of the face. The left upper limb was completely paralyzed. The biceps and triceps reflexes were exaggerated on the left side and were prompt on the right side. The left lower limb was completely paralyzed except at the hip joint. The left limbs were not flaccid. The patellar reflex was exaggerated on the left side, and normal on the right. Ankle clonus and the Babinski reflex were obtained on the left side, but not on the right. Sensation was normal everywhere. The voluntary power of the right upper and lower limbs was normal.

Dr. Spiller stated that he had asked Dr. Maier to show this case as being one of unusual interest, inasmuch as there was a question as to whether there was a peripheral facial palsy on each side or whether it was central in origin. He was inclined to think it was central, although of the peripheral type. The man very clearly had had two cerebral attacks, after which the bilateral facial palsy had developed. When Dr. Spiller had first seen the patient the facial palsy was very marked on the right side, although there had since been some improvement on this side, and to some extent on the left. He stated that he first tested the electrical reactions in December, a few weeks after the development of the palsy, and got prompt response over the seventh nerves and their muscles, and while this was not a positive proof that the palsy was central, it was at least indicative of it. He believed that the man had had a small lesion in the left side of the brain at the knee of the internal capsule which involved the fibers for the face, and that he had since regained, in part, the power in the right side of the face. Later he had had a complete lesion involving the internal capsule on the right side. He thought it would be difficult to consider this a peripheral facial palsy, even though the upper part of the face was much involved. The implication of the upper part of the face could be explained by a lesion on each side of the brain cutting the fibers innervating the upper part of the face on each side. The complete hemiplegia on the left side, the prompt reaction to the faradic current in the face and the deviation of the tongue to the left, all led him to the belief that the palsy was of central origin. He thought the reason there were no bulbar symptoms in this case was because fibers controlling the muscles of the throat may be separate from those for the face and need not be involved when those for the face are involved.

Dr. Dercum thought Dr. Spiller's explanation an ingenious and a possible one, but believed there was also a lesion here nuclear in character. He thought it possible that the man had a polioencephalitis.

Dr. Gordon did not know of any reported case of complete hemiplegia having this involvement of the face. From the history he believed all the symptoms could be accounted for by hemorrhage in the pons. He thought the assumption of a bulbar origin would explain all the symptoms that the man presented.

Dr. Pickett referred to a case at the Medico-Chirurgical Hospital, of hemiplegia, doubtless embolic in character, which showed the peripheral facial type of palsy as a part of the hemiplegia. This disappeared in three weeks. He did not think it a rare condition. He thought that if this palsy were central, the bilateral innervation would not be operative, and we would get peripheral palsy on both sides.

Dr. Weisenburg stated that he had at first thought that the man had a peripheral facial palsy on the right side, but later that he had a hemorrhage in the right internal capsule causing left hemiplegia.

Dr. Mills thought Dr. Spiller's explanation quite possible, and the true explanation.

Dr. Spiller, in replying, stated that he did not see how the lesion could have been in the pons. To assume that it was located there would imply that the lesion had involved both facial nuclei and the right pyramidal tract, above the decussation, but the facial palsy on the right side was less severe than on the left side, and yet the hemiplegia was on the left side. It could hardly be supposed that such a lesion could avoid the left pyramidal tract in the pons, which it must have done if it were in the pons. The marked implication of the upper part of the face on each side could only be explained by a lesion on each side of the brain in the fibers for the innervation of this part of the face, or by peripheral palsy. The latter seemed improbable. If the paralysis on either side of the face was caused by a lesion in the facial nerve, the marked paralysis of the upper part of the face on the other side could not be explained by a central lesion.

A Case with Lesion in the Conus Medullaris.—This was exhibited by Dr. Howard M. Fussell by invitation.

Dr. Eshner thought it possible that there were multiple lesions in this case, and in that way the lumbar segments of the cord would be affected and the knee jerks lost.

A Case of Obscure Functional Spasm with Myotonia.—This was exhibited by Dr. F. X. Dercum.

Dr. Eshner thought the case might belong to one of the occupational neuroses. It seemed that a variety of movements induced the spasm just as repeated movements resulted in other occupational diseases, or as repeated efforts at writing cause scrivener's palsy.

A Case of Huntington's Chorea and a Case of Cerebral Diplegia.—These were exhibited by Dr. Van Gasken.

Unilateral and Bilateral Convulsions in Apoplexy.—Dr. William G. Spiller made some remarks on this subject.

Dr. Mills thought this subject quite interesting from a physiological as well as a clinical point of view, and that it is perfectly true, as Dr. Spiller had said, that usually convulsions do not occur in cases of hemorrhage that destroy the internal capsule. He said that it was the common experience in a multitude of cases of paralysis due to hemorrhage from Charcot's artery of cerebral hemorrhage that in the majority of cases convulsions are not present, although they are occasionally met with, as is also tonic spasticitv. He stated that physiological experiments had indicated that the pyramidal tracts when separated from their centers are irritable. In one of the earliest of the American experiments, which is classical in the history of localization, the current was first applied to the cortex, and then a flap of the cortex was turned up and irritation again applied, and similar results were obtained in both cases. Dr. Mills thought it could not be decided that the pyramidal tracts are not irritable.

Antero-Retrograde Amnesia.—Dr. Alfred Gordon read a paper on this subject.

Two Cases of Syringomyelia with Necropsy.—This paper was read by invitation by Mr. H. S. Hutchinson.

Dr. Weisenburg stated that he had reported a case of syringomyelia with choked disc, his case being the third on record. Hydrocephalus had been the cause of the choked disc. He said that in no such case had the diagnosis of syringomyelia been made during life.

Dr. Mills referred to a case under his care which presented symptoms of syringomyelia in the upper extremities and to a certain extent in the lower extremities. The case had spastic contractures in the lower extremities with paraplegia, exaggerated reflexes, and had also the beginning irritative phenomena in the lower extremities, pain in the limbs and back, and recently pain in the trunk and upper extremities. He had concluded, however, that the proper diagnosis was pachymeningitis.

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 27, 1906.

The President, Dr. D. J. McCarthy, in the Chair.

A Case of Huntington's Chorea, and a Case of Lead Paralysis of the Upper Arm Type.—These were exhibited by Dr. Charles S. Potts.

Two Cases of Korsakoff's Psychosis with Recovery.—These were reported by Dr. T. H. Weisenburg.